

BILATERAL SPONTANEOUS AND SIMULTANEOUS RUPTURE OF THE QUADRICEPS TENDONS IN GOUT

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A fifty-four-year-old retired business man, born in Iran, was admitted to the orthopaedic department after he suddenly collapsed for no apparent reason while walking down the street and was unable to stand up again. At the time of admission both his knees were swollen with haematomata above the patellae. There were wide gaps where the quadriceps tendons should have been, and the patient could not actively extend his knees. Radiographs showed both patellae to be displaced, a finding which corresponds with tears of both quadriceps tendons (Fig. 1).

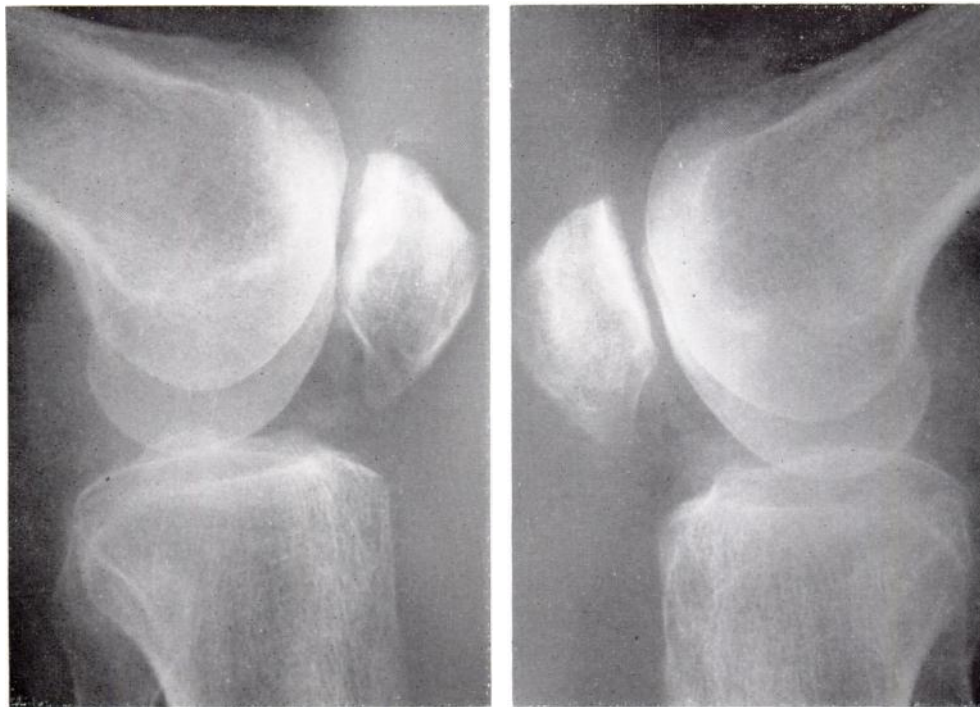


FIG. 1

Radiograph of the knees showing the downward displacement of both patellae.

The patient had been suffering from chronic renal disease for the last four years. He had been investigated in hospital where high blood urea values were found and renal biopsy showed chronic interstitial nephritis and arteriosclerotic changes. He was put on a Giovanetti low protein diet. During the last year he had also complained of pain and swelling in his knees and feet, for which he had received physiotherapy.

On admission the blood urea values were between 150 and 200 milligrams per cent. Traces of protein and many leucocytes were found in the urine. Blood pressure was 140/80 millimetres of mercury. Intravenous pyelography showed both kidneys smaller than normal.

Operation—At operation transverse tears of both quadriceps tendons were found. Both tendons appeared degenerate, with many small palpable nodules about two millimetres in diameter. The tendons were repaired by the technique of Scuderi (1958).

Histological examination—Histological examination of the synovial tissue from both sides revealed extensive fibrinoid necrosis and fibrin deposits. There was marked synovial

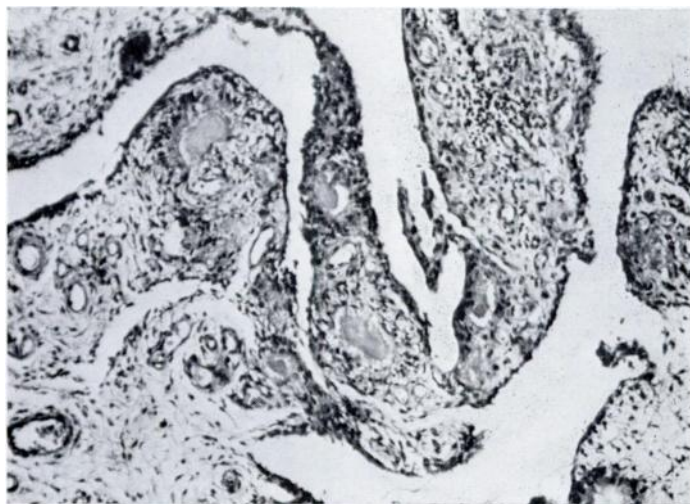


FIG. 2

Chronic gouty synovitis with villous proliferation, chronic inflammatory and granulomatous formations. (Haematoxylin and eosin $\times 40$.)

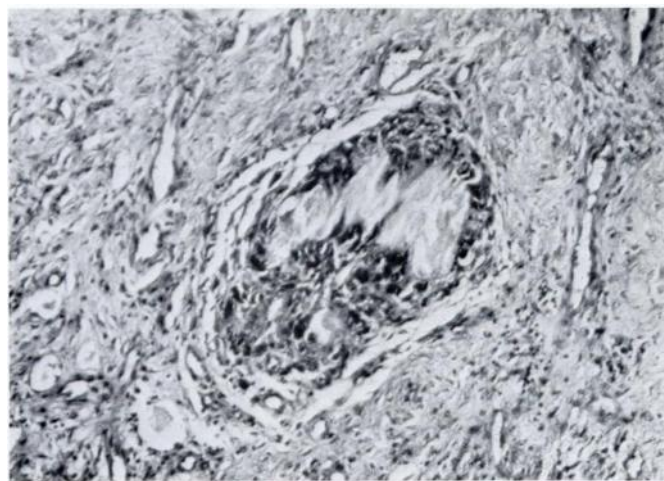


FIG. 3

Chronic gouty arthritis with tophus in the synovial tissue. The needle-like spaces probably represent uric acid crystals which dissolved during fixation. (Haematoxylin and eosin $\times 100$.)

proliferation with formation of villous processes infiltrated by lymphocytes, macrophages and plasma cells (Fig. 2). Numerous granuloma-like formations were seen (Figs. 3 to 5). These consisted of an amorphous eosinophilic centre in which slender needle-like spaces were present, sometimes arranged in parallel bundles, sometimes concentrically. Around these accumulations a marked inflammatory reaction comprised mainly of fibroblasts and giant cells was evident.

Because the material was formalin fixed, we were unable to prove the nature of the crystalline material which presumably had occupied the spaces within the granulomas and dissolved during fixation. A tentative diagnosis of gouty arthritis was made.

Further studies—Further blood examinations showed the uric acid level to be 14.9, 15.2 and 13.7 milligrams per cent (normal values 2–6 milligrams per cent). The level of uric acid in

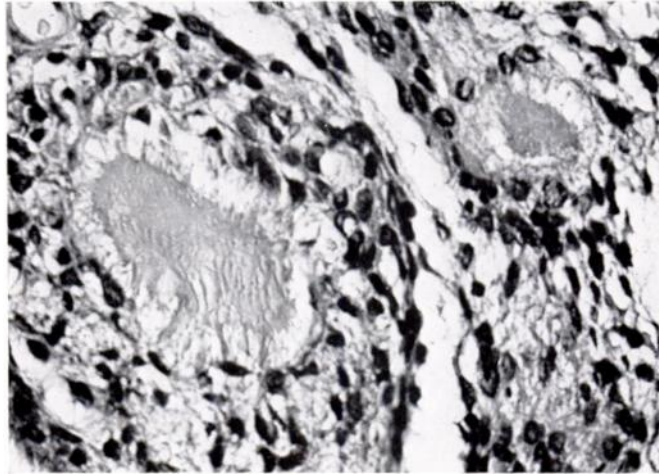


FIG. 4

Chronic gouty arthritis with tophus in the synovial tissue. (Haematoxylin and eosin · 400.)

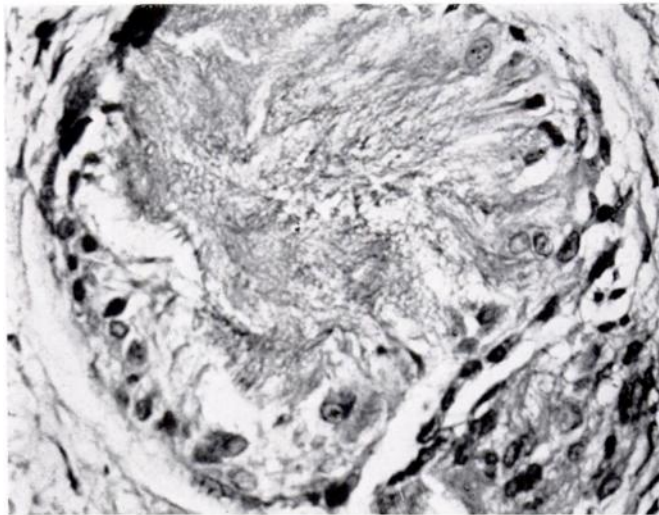


FIG. 5

Chronic gouty arthritis with tophus in the synovial tissue. (Haematoxylin and eosin · 400.)

the knee fluid, obtained by needle aspiration, was 15.9 milligrams per cent. Excretion of uric acid in the urine in twenty-four hours was 135 milligrams (normal 400–500).

On radiographic examination of the knees and first metatarsal heads small areas of transradiancy were seen near the joints. There were tophi in the patient's ear lobes.

Four months after operation the patient was walking well with the aid of a stick.

DISCUSSION

Bilateral spontaneous and simultaneous rupture of the quadriceps tendons is rare. Only four such cases have been described previously (Steiner and Palmer 1949, Wetzler and Merkow 1950, Preston and Adicoff 1962, Dalal and Whittam 1966). The ruptures are ascribed to degenerative changes causing weakening of the tendon tissue. Preston and Adicoff (1962) thought that deposits of calcium were the cause of the weakening in the case they reported of a patient suffering from hyperparathyroidism. They also stated that because of the rarity of bilateral spontaneous rupture, they first diagnosed it as being due to an inflammatory process, such as atypical rheumatoid arthritis, mainly because of the swelling, heat, pain and elevated sedimentation rate, and the absence of any clear history of injury.

To the best of our knowledge no case of tendon rupture caused by gout has yet been described. Our patient most probably was suffering from gout before the rupture took place. The weakening of the tendons was caused by fibrinoid necrosis and chronic inflammatory reaction, which not infrequently accompany chronic tophaceous synovitis. These degenerative changes gradually affected his quadriceps tendons until the simple strain of walking tore one of them, and the patient's attempt to prevent himself from falling tore the other. Renal biopsy did not show any changes specific for gout, but the changes found are known to accompany gout in a high percentage of cases (Sokoloff 1957). The renal findings do not exclude gout nephrosis as a possible cause for the chronic renal failure in this case.

SUMMARY

1. A case of bilateral spontaneous and simultaneous rupture of the quadriceps tendons is described.
2. The underlying cause was found to be gouty affection of the tendons.
3. So far as is known, a similar case has not previously been recorded.

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